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Relationship between edema and wall thickness in acute myocardial infarction

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Introduction

Experimental studies using echocardiography have shown that reperfusion of acutely infarcted myocardium induces changes in end-diastolic wall thickness (EDWT), likely caused by myocardial edema. However, the relationship between edema and EDWT has not been described.

Purpose

The purpose of this study is to assess the relationship between regional EDWT and myocardial edema as defined by T2-weighted CMR in patients with reperfused acute myocardial infarction (MI).

Methods

We studied nineteen patients with reperfused acute MI without microvascular obstruction (LAD: n = 7; LCX: n = 1; RCA: n = 11). We also studied six healthy subjects. Patients underwent cine CMR imaging, T2-weighted CMR (STIR), and late gadolinium enhancement (LGE) CMR 2.5 +/- 1.2 days after acute MI. Each short axis was divided into 12-equi-angular segments. The presence of edema and infarction was verified and evaluated semi-quantitatively for each segment, using 2SD above the mean of normal myocardium as a threshold on T2-weighted CMR and 5SD above the mean on LGE images. Wall thickness was measured in the salvaged area at risk, the infarction and in region without evidence for edema using a centerline method with 48 chords (Figure 1). We used the mean of 4 chords as EDWT, resulting in a total of 12 segments.

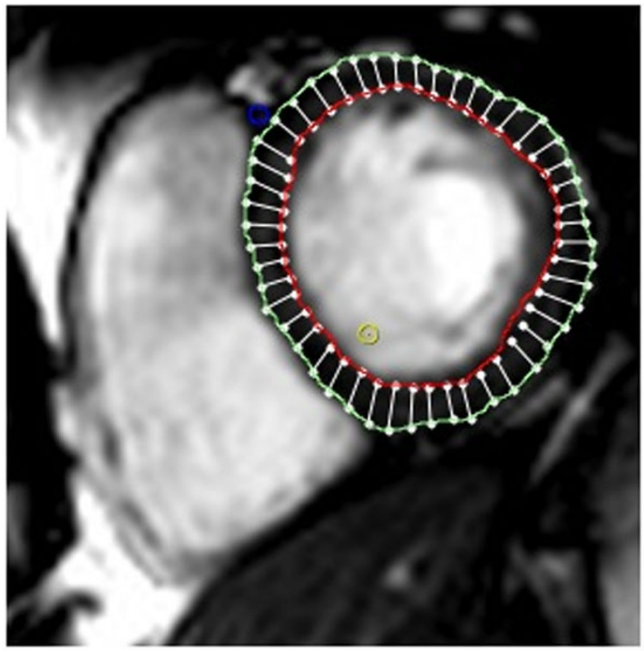

Figure 1
Measurement of end-diastolic wall thickness (EDWT). The wall thickness was measured uesing a centerline method with 48 chords. We used the mean of 4 chords as EDWT, resulting in a total or 12 segments.

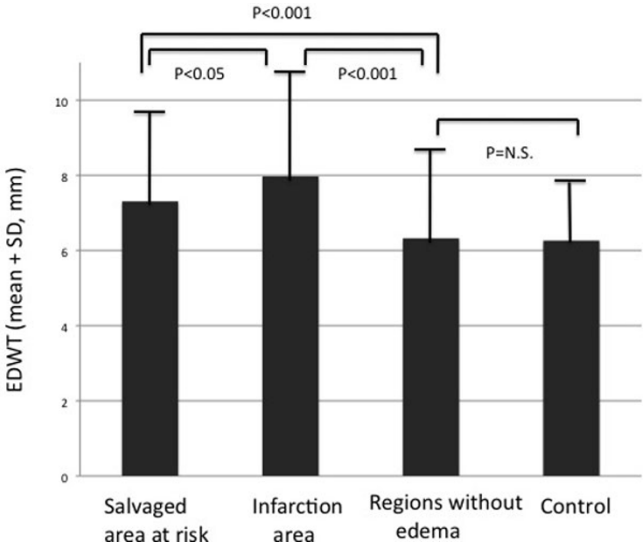

Figure 2
End-diastolic wall thickness (EDWT) of each area. The salvaged area at risk and infarction area had significantly increased EDWT compared with regions with edema. Infarction area had significantly increased EDWT compared with salvaged are at risk.

Results

972 segments (81 slices) from 19 patients and 372 segments (31 slices) from healthy subjects were analyzed. Segmental EDWT of regions without edema was not significantly different from that of healthy subjects (6.32 +/- 2.22 mm and 6.26 +/- 1.73 mm, p = N.S.). Edema was observed in 358 segments and infarction was observed in 95 segments. The salvaged area at risk had significantly increased EDWT compared with regions without edema (7.31 ± 2.39 mm and 6.32 ± 2.22 mm, $p < 0.05$, respectively). The infarction area also had significantly increased EDWT compared with area without edema (7.97 ± 2.48 mm, $p < 0.05$). The infarcted segments had a significantly increased EDWT compared to the salvaged area at risk ($p < 0.05$, Figure 2). In healthy subjects, EDWT in the LCX area was less than in the RCA area (5.89 +/- 1.72 mm and 6.63 +/- 1.71 mm, $p < 0.05$). In an additional analysis excluding LCX area (648 segments), the salvaged area at risk had an increased EDWT compared with regions without edema (7.46 +/- 2.46 mm and 6.93 +/- 2.46 mm, $p < 0.05$, respectively) and also had less EDWT when compared to infarcted myocardium (8.24 +/- 2.40 mm, $p < 0.05$).

Conclusion

Myocardial edema as defined by T2-weighted CMR is strongly associated with regionally increased EDWT. Within the area at risk, infarcted segments have a significantly increased EDWT compared to edematous, yet salvaged myocardium.